

## **Part IV-B: Table of HIV Monoclonal Antibodies and Polyclonal Antibody Responses**

**All HIV MAbs and polyclonal Abs that bind to linear epitopes 30 amino acids or less in length arranged by protein position. Abs that bind to conformational epitopes are listed at the end of each protein section. The table entries have been sorted in a nested way, first by protein, then by HXB2 start location, then by HXB2 end location, then by antibody type, and finally by antibody name. Any antibodies whose HXB2 location is unknown will appear at the end of the listing of the protein in which they are located.**

Table of HIV MAbs

Table 1: p17

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
1 L14.17	p17(11–25)	p17(11–25 BRU)	GELDRWEKIRLRPGG	no	Vaccine	murine(IgG)
	<p><b>Vaccine:</b> <i>Vector/type:</i> viral lysate    <i>Strain:</i> BRU    <i>HIV component:</i> virus  <b>References:</b> [Tatsumi (1990), Robert-Hebmann (1992b), Robert-Hebmann (1992a)]</p>					
2 polyclonal	p17(11–25)	p17(11–25 LAI)	GELDRWEKIRLRPGG	no	Vaccine	mouse( )
	<p><b>Vaccine:</b> <i>Vector/type:</i> recombinant protein, virus-like particle    <i>Strain:</i> LAI    <i>HIV component:</i> p24, p17, p55    <i>Stimulatory Agents:</i> Freund's adjuvant  <b>References:</b> [Truong (1997)]</p> <ul style="list-style-type: none"> <li>• An ELISA assay was used to study a panel of Gag peptides – mature p24 CA epitopes mapped to residues 176–192, 201–218, 233–253, 285–304, and were recognized by antibodies elicited by rp24CA – one p17MA epitope, residues 11–25, and one p24CA epitope, residues 176–192, were recognized by antibodies raised against anti-p55 virus-like particles, suggesting different antigenic properties for p24CA and p17MA antibodies depending on whether they are produced against the mature soluble protein or the immature assembled form of the Gag protein [Truong (1997)]</li> </ul>					
3 32/5.8.42	p17(dis 12–19 + 100–105)	p17(dis 12–19 IIIB)	ELDRWEKI + ALDKIE		Vaccine	murine(IgG)
	<p><b>Vaccine:</b> <i>Vector/type:</i> viral lysate  <b>References:</b> [Papsidero (1989)]</p> <ul style="list-style-type: none"> <li>• 32/5.8.42: Binds to two discontinuous regions, positions 12–19 and 100–105, peptides ELDRWEKI and ALDKIE – inhibited infectivity of cell free virus [Papsidero (1989)]</li> </ul>					
4 HyHIV-1	p17(12–29)	p17(12–29 JMH1)	ELDKWEKIRLRPGGKTLY		Vaccine	murine(IgG1)
	<p><b>Vaccine:</b> <i>Vector/type:</i> recombinant protein    <i>HIV component:</i> p17  <b>References:</b> [Liu (1995), Ota &amp; Ueda(1998)]</p> <ul style="list-style-type: none"> <li>• HyHIV-1: This paper compares the results of affinity constant (<math>K_a</math>) measurements of anti-p17 MAbs using double Ab methods versus the faster, isotope-free BIAcore system, and results were found to be similar for HyHIV-(1–6), six MAbs which all bind to the first alpha helix of p17, a functional domain for both membrane binding and nuclear localization [Ota &amp; Ueda(1998)]</li> </ul>					
5 HyHIV-2	p17(12–29)	p17(12–29 JMH1)	ELDKWEKIRLRPGGKTLY	no	Vaccine	murine(IgG1)
	<p><b>Vaccine:</b> <i>Vector/type:</i> recombinant protein    <i>HIV component:</i> p17  <b>References:</b> [Liu (1995), Ota &amp; Ueda(1998)]</p> <ul style="list-style-type: none"> <li>• HyHIV-2: This paper compares the results of affinity constant (<math>K_a</math>) measurements of anti-p17 MAbs using double Ab methods versus the faster, isotope-free BIAcore system, and results were found to be similar for HyHIV-(1–6), six MAbs which all bind to the first alpha helix of p17, a functional domain for both membrane binding and nuclear localization [Ota &amp; Ueda(1998)]</li> </ul>					

Table of HIV MABs

6	HyHIV-3	p17(12–29)	p17(12–29 JMH1)	ELDKWEKIRLRPGGKTLY	no	Vaccine	murine(IgG1)
	<b>Vaccine:</b>	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> p17			
		<b>References:</b> [Liu (1995), Ota & Ueda(1998)]					
		<ul style="list-style-type: none"> <li>HyHIV-3: This paper compares the results of affinity constant (<math>K_a</math>) measurements of anti-p17 MABs using double Ab methods versus the faster, isotope-free BIAcore system, and results were found to be similar for HyHIV-(1–6), six MABs which all bind to the first alpha helix of p17, a functional domain for both membrane binding and nuclear localization [Ota &amp; Ueda(1998)]</li> </ul>					
7	HyHIV-4	p17(12–29)	p17(12–29 JMH1)	ELDKWEKIRLRPGGKTLY?	no	Vaccine	murine(IgG1)
	<b>Vaccine:</b>	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> p17			
		<b>References:</b> [Liu (1995), Ota (1998), Ota & Ueda(1998)]					
		<ul style="list-style-type: none"> <li>HyHIV-4: epitope uncertain, based on the best estimate from JMH1 sequence– <math>K_a</math> is <math>1.8 \times 10^7 \text{ M}^{-1}</math> for rec p17 – stains the surface of infected cells indicating the antigen is exposed at the cell surface [Ota (1998)]</li> <li>HyHIV-4: This paper compares the results of affinity constant (<math>K_a</math>) measurements of anti-p17 MABs using double Ab methods versus the faster, isotope-free BIAcore system, and results were found to be similar for HyHIV-(1–6), six MABs which all bind to the first alpha helix of p17, a functional domain for both membrane binding and nuclear localization [Ota &amp; Ueda(1998)]</li> </ul>					
8	HyHIV-5	p17(12–29)	p17(12–29 JMH1)	ELDKWEKIRLRPGGKTLY	no	Vaccine	murine(IgG1)
	<b>Vaccine:</b>	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> p17			
		<b>References:</b> [Liu (1995), Ota & Ueda(1998)]					
		<ul style="list-style-type: none"> <li>HyHIV-5: This paper compares the results of affinity constant (<math>K_a</math>) measurements of anti-p17 MABs using double Ab methods versus the faster, isotope-free BIAcore system, and results were found to be similar for HyHIV-(1–6), six MABs which all bind to the first alpha helix of p17, a functional domain for both membrane binding and nuclear localization [Ota &amp; Ueda(1998)]</li> </ul>					
9	HyHIV-6	p17(12–29)	p17(12–29 JMH1)	ELDKWEKIRLRPGGKTLY	no	Vaccine	murine(IgG1)
	<b>Vaccine:</b>	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> p17			
		<b>References:</b> [Liu (1995), Ota & Ueda(1998)]					
		<ul style="list-style-type: none"> <li>HyHIV-6: This paper compares the results of affinity constant (<math>K_a</math>) measurements of anti-p17 MABs using double Ab methods versus the faster, isotope-free BIAcore system, and results were found to be similar for HyHIV-(1–6), six MABs which all bind to the first alpha helix of p17, a functional domain for both membrane binding and nuclear localization [Ota &amp; Ueda(1998)]</li> </ul>					
10	32/1.24.89	p17(17–22)	p17(17–22 IIIB)	EKIRLR	L	Vaccine	murine(IgG)
	<b>Vaccine:</b>	<i>Vector/type:</i> viral lysate					
		<b>References:</b> [Papsidero (1989)]					
		<ul style="list-style-type: none"> <li>32/1.24.89: Inhibited infectivity of cell free virus [Papsidero (1989)]</li> </ul>					

**Table of HIV MAbs**

11	3B10	p17(19–38)	p17(19–38 SIVmac)	IRLPGGKKKYMLKHVVWAA	no Vaccine	murine(IgG1)
<p><b>Vaccine:</b> <i>Vector/type:</i> inactivated virus    <i>Strain:</i> AGM TYO-7    <i>HIV component:</i> virus</p> <p><b>References:</b> [Otteken (1992)]</p> <ul style="list-style-type: none"> <li>• 3B10: Recognized an epitope present on HIV-2/SIVmac (MAC251/32H) , SIVagm, HIV-1, and SIVmnd, demonstrating that the matrix protein of all nine HIV and SIV isolates tested in this study expresses at least one conserved immunogenic epitope recognized serologically [Otteken (1992)]</li> </ul>						
12	3E11	p17(19–38)	p17(19–38 SIVmac)	IRLPGGKKKYMLKHVVWAA	no Vaccine	murine(IgG1)
<p><b>Vaccine:</b> <i>Vector/type:</i> inactivated virus    <i>Strain:</i> AGM TYO-7    <i>HIV component:</i> virus</p> <p><b>References:</b> [Otteken (1992), Nilsen (1996)]</p> <ul style="list-style-type: none"> <li>• 3E11: There is another MAb with this ID that recognizes integrase [Nilsen (1996)]</li> <li>• 3E11: Recognized an epitope present on HIV-2/SIVmac (MAC251/32H), SIVagm, HIV-1, and SIVmnd, demonstrating that the matrix protein of all nine HIV and SIV isolates tested in this study expresses at least one highly conserved immunogenic epitope [Otteken (1992)]</li> </ul>						
13	8H10	p17(30–52)	p17(30–52 JMH1)	KLKHIVWASRELERFAVNPGL-LE	Vaccine	murine(IgM)
<p><b>Vaccine:</b> <i>Vector/type:</i> peptide    <i>Strain:</i> JMH-1    <i>HIV component:</i> p17    <i>Stimulatory Agents:</i> BSA</p> <p><b>References:</b> [Ota (1999), Ota &amp; Ueda(1999)]</p> <ul style="list-style-type: none"> <li>• 8H10: This p17 MAb also can bind to the V3 loop [Ota (1999)]</li> <li>• 8H10: Inhibits viral replication of the HIV-1 infected MT-4 cells by decreasing p17 protein levels in the infected cells, and the effect of growing the 8H10 hybridoma in co-culture with HIV-1 infected MT-4 cells was studied [Ota &amp; Ueda(1999)]</li> </ul>						
14	HyHIV-21	p17(30–52)	p17(30–52 JMH1)	KLKHIIWASRELERFAVNPGLLE	no Vaccine	murine(IgG2a)
<p><b>Vaccine:</b> <i>Vector/type:</i> recombinant protein    <i>HIV component:</i> p17</p> <p><b>References:</b> [Liu (1995), Ota (1998)]</p> <ul style="list-style-type: none"> <li>• HyHIV-21: epitope uncertain, based on the best estimate from JMH1 sequence – <math>K_a</math> is <math>3.6 \times 10^6 \text{ M}^{-1}</math> for rec p17 – stains the surface of infected cells indicating the antigen is exposed at the cell surface –inhibited growth of HIV-1 JMH1 in MT-4 cells when added 24 hours after the initial culture [Ota (1998)]</li> </ul>						
15	B4f8	p17(51–65)	p17(51–65)	LETSEGCRQILGQLQ	no Vaccine	rat(IgG2a)
<p><b>Vaccine:</b> <i>Vector/type:</i> infected-cell lysate    <i>Strain:</i> IIIB    <i>HIV component:</i> virus</p> <p><b>References:</b> [Shang (1991)]</p> <ul style="list-style-type: none"> <li>• B4f8: Did not bind live infected cells, only cells that had been made permeable with acetone [Shang (1991)]</li> </ul>						

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16	HyHIV-22	p17(52–83)	p17(53–87 JMH1)	ETSEGCRQILGQRQPSLQTGS- EELRSLYNTIH?	no Vaccine	murine(IgG1)
	<b>Vaccine:</b>	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> p17		
		<b>References:</b> [Liu (1995), Ota (1998)]				
		<ul style="list-style-type: none"> <li>HyHIV-22: stains the surface of infected cells indicating the antigen is exposed at the cell surface – <math>K_a</math> is <math>2.3 \times 10^5 \text{ M}^{-1}</math> for rec p17 [Ota (1998)]</li> </ul>				
17	12H-D3b3	p17(62–78)	p17(62–78)	GQLQPSLQTGSEELRSL	no Vaccine	rat(IgG2a)
	<b>Vaccine:</b>	<i>Vector/type:</i> infected-cell lysate		<i>Strain:</i> IIIB	<i>HIV component:</i> virus	
		<b>References:</b> [Shang (1991)]				
		<ul style="list-style-type: none"> <li>12H-D3b3: Did not bind live infected cells, only cells that had been made permeable with acetone [Shang (1991)]</li> </ul>				
18	12G-A8g2	p17(86–115)	p17(86–115)	YCVHQRIEIKDTKEALDKIEE- EQNKSKKKA	no Vaccine	rat(IgG2a)
	<b>Vaccine:</b>	<i>Vector/type:</i> infected-cell lysate		<i>Strain:</i> IIIB	<i>HIV component:</i> virus	
		<b>References:</b> [Shang (1991)]				
		<ul style="list-style-type: none"> <li>12G-A8g2: Bound to 30-mer, but not to internal peptides – did not bind live infected cells – antigenic domain known as HPG30 [Shang (1991)]</li> </ul>				
19	12G-D7h11	p17(86–115)	p17(86–115)	YCVHQRIEIKDTKEALDKIEE- EQNKSKKKA	no Vaccine	rat(IgG2a)
	<b>Vaccine:</b>	<i>Vector/type:</i> infected-cell lysate		<i>Strain:</i> IIIB	<i>HIV component:</i> virus	
		<b>References:</b> [Shang (1991)]				
		<ul style="list-style-type: none"> <li>12G-D7h11: Bound to 30-mer, but not to internal peptides – did not bind live infected cells – antigenic domain known as HPG30 [Shang (1991)]</li> </ul>				
20	12G-H1c7	p17(86–115)	p17(86–115)	YCVHQRIEIKDTKEALDKIEE- EQNKSKKKA	no Vaccine	rat(IgG)
	<b>Vaccine:</b>	<i>Vector/type:</i> infected-cell lysate		<i>Strain:</i> IIIB	<i>HIV component:</i> virus	
		<b>References:</b> [Shang (1991)]				
		<ul style="list-style-type: none"> <li>12G-H1c7: Bound to 30-mer, but not to internal peptides – did not bind live infected cells – antigenic domain known as HPG30 [Shang (1991)]</li> </ul>				
21	12I-D12g2	p17(86–115)	p17(86–115)	YCVHQRIEIKDTKEALDKIEE- EQNKSKKKA	no Vaccine	rat(IgG2a)
	<b>Vaccine:</b>	<i>Vector/type:</i> infected-cell lysate		<i>Strain:</i> IIIB	<i>HIV component:</i> virus	
		<b>References:</b> [Shang (1991)]				

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- 12I-D12g2: Bound to 30-mer, but not to internal peptides – did not bind live infected cells – antigenic domain known as HPG30 [Shang (1991)]

22	polyclonal	p17(86–115)	p17(86–115)	YSVHQRIDVKDTKEALEKIEE- EQNKSKKKA	L Vaccine	murine(IgA)
<p><b>Vaccine:</b> <i>Vector/type:</i> peptide    <i>Strain:</i> Multiple B-clade    <i>HIV component:</i> V3 loop, CD4BS, HPG30    <i>Stimulatory</i>  <i>Agents:</i> cholera toxin adjuvant</p> <p><b>References:</b> [Bukawa (1995)]</p> <ul style="list-style-type: none"> <li>• Polyclonal secretory IgA antibody raised by oral mucosal immunization is able to neutralize IIIB, SF2, and MN – HIV-1 neutralization may be due to the V3, CD4 or HPG30 component of the multicomponent peptide immunogen [Bukawa (1995)]</li> </ul>						
23	HyHIV-15	p17(87–115)	p17(87–115 JMH1)	SVHQRIDVKDTKEALEKIEE- EQNKSKKKA?	L Vaccine	murine(IgG1)
<p><b>Vaccine:</b> <i>Vector/type:</i> recombinant protein    <i>HIV component:</i> p17</p> <p><b>References:</b> [Liu (1995), Ota (1998)]</p> <ul style="list-style-type: none"> <li>• HyHIV-15: epitope uncertain, based on the best estimate from JMH1 sequence – <math>K_a</math> is <math>1.4 \times 10^7 \text{ M}^{-1}</math> for rec p17 – stains the surface of infected cells indicating the antigen is exposed at the cell surface – inhibited growth of HIV-1 JMH1 in MT-4 cells when added 24 hours after the initial culture [Ota (1998)]</li> </ul>						
24	32/5.8.42	p17(dis 12–19 + 100–105)	p17(dis IIIB)	ELDRWEKI + ALDKIE	no Vaccine	murine(IgG)
<p><b>Vaccine:</b> <i>Vector/type:</i> viral lysate    <i>HIV component:</i> virus</p> <p><b>References:</b> [Papsidero (1989)]</p> <ul style="list-style-type: none"> <li>• 32/5.8.42: Inhibited infectivity of cell free virus – bound to two peptides, ELDRWEKI and ALDKIE, at positions 12–19 + 100–105 [Papsidero (1989)]</li> </ul>						
25	11H9	p17(101–115)	p17(101–115 SF2)	LEKIEEEQNKSKKKA?	Vaccine	murine(IgG1)
<p><b>Vaccine:</b> <i>Vector/type:</i> inactivated virus    <i>Strain:</i> CBL-1    <i>HIV component:</i> virus</p> <p><b>Donor:</b> R. B. Ferns and R. S. Tedder</p> <p><b>References:</b> [Ferns (1987), Ferns (1989)]</p> <ul style="list-style-type: none"> <li>• 11H9: Reactive against p18 and p55 [Ferns (1987)]</li> <li>• 11H9: UK Medical Research Council AIDS reagent: ARP344</li> </ul>						
26	3-H-7 (3H7)	p17(113–122)	p17(113–122 BH10)	KKAQQAAADT	L Vaccine	murine(IgG)
<p><b>Vaccine:</b> <i>Strain:</i> IIIB</p> <p><b>References:</b> [Niedrig (1989), Robert-Hebmann (1992b), Robert-Hebmann (1992a), Levin (1997)]</p> <ul style="list-style-type: none"> <li>• 3-H-7: No cross-reactivity with HIV-2 ROD or SIV MAC by immunoblot [Niedrig (1989)]</li> </ul>						

- 3-H-7: Called 3H7 – using a bicistronic vector, an intracellular Fab intrabody, 3H7, can inhibit HIV-1 infection when expressed in the cytoplasm of dividing CD4+ T cells – HXBIIIIB and SI primary isolate virions from 3H7 expressing cells were far less infectious – 3H7 intrabody acts both at the stage of nuclear import and virus particle assembly [Levin (1997)]

27	C5126	p17(113–122)	p17(113–122 HXB2)	KKAQQAADT	no Vaccine	murine(IgG1 $\kappa$ )
<p><b>Vaccine:</b> <i>Vector/type:</i> viral lysate    <i>HIV component:</i> virus</p> <p><b>References:</b> [Hinkula (1990)]</p> <ul style="list-style-type: none"> <li>• C5126: Defined epitope by peptide blocking of binding to native protein – WB reactive with p53 and p17 [Hinkula (1990)]</li> </ul>						
28	1D9	p17(119–132)	p17(121–134 SF2)	AAGTGNSSQVSQNY	Vaccine	murine(IgG2a)
<p><b>Vaccine:</b> <i>Vector/type:</i> inactivated virus    <i>Strain:</i> CBL-1    <i>HIV component:</i> virus</p> <p><b>Donor:</b> R. B. Ferns and R. S. Tedder</p> <p><b>References:</b> [Ferns (1987), Ferns (1989)]</p> <ul style="list-style-type: none"> <li>• 1D9: Reactive against p18, but not p55 [Ferns (1987)]</li> <li>• 1D9: UK Medical Research Council AIDS reagent: ARP316</li> </ul>						
29	4C9	p17(119–132)	p18(121–134 SF2)	AAGTGNSSQVSQNY	Vaccine	murine(IgG2a)
<p><b>Vaccine:</b> <i>Vector/type:</i> inactivated virus    <i>Strain:</i> CBL-1    <i>HIV component:</i> virus</p> <p><b>Donor:</b> R. B. Ferns and R. S. Tedder</p> <p><b>References:</b> [Ferns (1987), Ferns (1989)]</p> <ul style="list-style-type: none"> <li>• 4C9: Reactive against p18, but not p55 [Ferns (1987)]</li> <li>• 4C9: UK Medical Research Council AIDS reagent: ARP342</li> </ul>						
30	4H2B1	p17(119–132)	p17(121–134 SF2)	AAGTGNSSQVSQNY		murine(IgG1)
<p><b>Donor:</b> R. B. Ferns and R. S. Tedder</p> <p><b>References:</b> [Ferns (1987), Ferns (1989)]</p> <ul style="list-style-type: none"> <li>• 4H2B1: Reactive against p18 and p55 of multiple isolates [Ferns (1987)]</li> <li>• 4H2B1: UK Medical Research Council AIDS reagent: ARP315</li> </ul>						
31	9G5	p17(119–132)	p17(121–134 SF2)	AAGTGNSSQVSQNY	Vaccine	murine(IgM)
<p><b>Vaccine:</b> <i>Vector/type:</i> inactivated virus    <i>Strain:</i> CBL-1    <i>HIV component:</i> virus</p> <p><b>Donor:</b> R. B. Ferns and R. S. Tedder</p> <p><b>References:</b> [Ferns (1987), Ferns (1989)]</p> <ul style="list-style-type: none"> <li>• 9G5: Reactive against p18, but not p55 [Ferns (1987)]</li> <li>• 9G5: UK Medical Research Council AIDS reagent: ARP343</li> </ul>						
32	15–21	p17(121–132)	p17(121–132 BRU)	DTGHSSQVSQNY	no Vaccine	murine(IgG)
<p><b>Vaccine:</b> <i>Strain:</i> BRU</p> <p><b>References:</b> [Robert-Hebmann (1992b), Robert-Hebmann (1992a)]</p>						

**Table of HIV MAbs**

33	31-11	p17(121-132)	p17(121-132 BRU)	DTGHSSQVSQNY	no Vaccine	murine(IgG)
	<b>Vaccine:</b>	<i>Strain:</i> BRU	<b>References:</b> [Robert-Hebmann (1992b), Robert-Hebmann (1992a)]			
34	sc-FV p17	p17(121-132)	p17(121-132 BRU)	DTGHSSQVSQNY	Vaccine	murine(IgG1 $\kappa$ )
	<b>Vaccine:</b>	<i>Strain:</i> BRU	<b>Ab type:</b> C-term <b>Donor:</b> Paul Zhou, NIH, Bethesda, MD, USA			
			<b>References:</b> [Robert-Hebmann (1992a), Tewari (1998)]			
			<ul style="list-style-type: none"> <li>• A single chain Ab (sc-FV) was made from an anti-p17 MAb, and intracellular binding of sc-FV resulted in inhibition of viral replication that was more pronounced when the sc-FV was expressed in the cytoplasm instead of the nucleus [Tewari (1998)]</li> </ul>			